A Dissertation on

SCREENING OF DIABETIC PATIENTS ATTENDING DIABETOLOGY OUT PATIENT DEPARTMENT FOR PRIMARY OPEN ANGLE GLAUCOMA

COIMBATORE MEDICAL COLLEGE HOSPITAL

Dissertation submitted in

Partial fulfilment of the regulations required for the award of

M.S. OPHTHALMOLOGY

BRANCH-III

APRIL - 2016

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 32, TAMIL NADU

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This is to certify that the dissertation entitled **" SCREENING OF DIABETIC PATIENTS ATTENDING DIABETOLOGY OUT PATIENT DEPARTMENT FOR PRIMARY OPEN ANGLE GLAUCOMA"** is a bonafide and research work done by **Dr. K. NITHYA** Postgraduate in M.S. Ophthalmology, Coimbatore Medical College & Hospital, Coimbatore under my direct guidance and supervision, to my satisfaction, in partial fulfilment of the regulations required for the award of M.S. Degree in Ophthalmology (Branch III) .

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Character count:	36,943
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This is submitted to The Tamil Nadu Dr. M.G.R. Medical University , Chennai in partial fulfilment of regulations required for the M.S. Ophthalmology, Branch III Degree Examination to be held in April 2016 .

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ACKNOWLEDGEMENT

I express my gratitude and sincere thanks to **Dr. A. EDWIN JOE MD , BL** Dean, Coimbatore Medical College for permitting me to do the thesis work .

I would like to express my gratitude to my guide and teacher **Dr. M. Hemanandini M.S, D.O**, Associate professor and Head of the Department of Ophthalmology for her valuable guidance and moral support to complete this dissertation work .

I also thank **Dr. S. VengoPrassadh MD, D.Diab**, Head Of Department and Associate professor of Diabetology, Coimbatore Medical College , Coimbatore for allowing me to do my dissertation in the patients attending diabetology out patient department .

I wish to extend my sincere gratitude to all my Assistant Professors **Dr. C. Jeevakala M.S, D.O, Dr. P. Sumathy M.S, Dr. J. Saravanan M.S, Dr. K. Malligai D.O, Dr. P. Mohanapriya M.S,** and **Dr. K.Sathya M.S,** in the department of ophthalmology, Coimbatore Medical College for the encouragement and for all that they have taught me.

Thanks to the administrators and nursing staff for having helped me whole heartedly to complete this work .

I am grateful to my parents, husband and child for giving me so much support and enlightening me throughout my life . I am grateful to all my colleagues, present and past, in the department of ophthalmology for being so friendly, helpful and co-operative, in preparing my dissertation .

Finally, I would like to express my sincere thanks to all my patients for their immense and kind co-operation.

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ABBREVIATIONS & ACRONYMS

POAG	-	Primary Open Angle Glaucoma
NTG	-	Normal Tension Glaucoma
IOP	-	Intra Ocular Pressure
CCT	-	Central Corneal Thickness
UCVA	-	Uncorrected Visual Acuity
BCVA	-	Best Corrected Visual Acuity
NCT	-	Non – Contact Tonometry
AT	-	Applanation Tonometry
DM	-	Diabetes Mellitus
I	-	Type I Diabetes Mellitus
II	-	Type II Diabetes Mellitus

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INTRODUCTION

Glaucoma (derived from the Greek word “glaukos” meaning blue hue) is one of the major public health problem, causing irreversible blindness. WHO statistics shows, the second commonest cause of blindness is glaucoma[1]. About 12 million people are affected by glaucoma in India and the majority of them are undiagnosed . It is a silent thief of vision [2] as it is asymptomatic till very advanced stage[3]. When the patient presents to the eye clinic with visual dysfunction the disease will be most often in advanced stage and the visual loss is irrecoverable[4].

DM is one of risk factor in open angle glaucoma of primary type. The incidence of diabetes is rising rapidly all over the world[1]. In India there are around 62 million people with diabetes, and is about 7.1% of adult people in India.[9] Every year in India around ten lakh diabetic patients are dying due to its complications. Indian Heart Association states that our country is the 'diabetes - capital of the world'. More than 109 million people will have diabetes mellitus in 2035.[8] Diabetes causes microangiopathy which affects smaller vessels .

It disrupts the auto regulation of the vessels in optic nerve head and retina [15]. Impaired blood flow to the surface of the optic nerve head can further aggravate glaucomatous optic neuropathic changes in the optic nerve head.[15]

Optic disc of the diabetics prone to glaucomatous changes at a lower IOP than the optic disc of normal people. Automated perimetry can detect the visual field defects only after 40% of nerve fibres are damaged. This is more important in diabetes patients who also have an additional risk factor of developing diabetic retinopathy which will further affect the visual acuity. Hence it is advisible to screen all the patients of diabetes mellitus to detect POAG in the early stage even when there is no visual complaints.

PRIMARY OPEN ANGLE GLAUCOMA

It is a chronic progressive optic neuropathy with acquired atrophy of the optic nerve and loss of retinal ganglion cells and their axons with intra - ocular pressure (IOP) being one of the risk factor, with the angle being open.[5].

Open angle glaucoma of primary type commonly occurs in adults above the age of forty years and more often there is a positive family history. It has multi – factorial inheritance and also polygenic[6] in nature. Developing open angle glaucoma of primary type risk in relatives (first degree relatives) is around 4–16%.[16 – 21] The Rotterdam study stated that there is a ten times high risk of developing glaucoma for the blood relations of patients having open angle glaucoma of primary type[22] .

HISTORY

As early as 10th century , Tabari in his "Hippocratic writings" had described inflammation of eye and raised intra - ocular tension . After that, understanding about glaucoma did not improve until the 1800s.

In 1709, Michel Brisseau was able to differentiate glaucoma from disorders of crystalline lens; he related glaucoma with vitreous. The belief continued until ophthalmoscopic optic nerve head examination was invented.

In 1818, Antoine-Pierre had described glaucoma well in detail and he is the first person who described colored halos around lights. In 1823, George J Guthrie had labelled the term "glaucoma."

In 1973 , William Hoyt gave first description of nerve fiber layer defects identified it as the earliest detectable sign of glaucoma.

With the advent of Automated Perimetry and optical coherence tomography, the diagnosis and management of glaucoma (POAG) made giant leaps forward.

PREVALENCE

At present , around sixty million people in the world are estimated to have glaucoma. Among them, about 11.2 million patients are from our country India [7, 8]. Every year more than two million population develop POAG in the world [9]. The estimated prevalence of Open Angle Glaucoma of primary type is 1.62% to 3.51%[8]. Its prevalence gradually increases with increasing age, with more than ninety percent are unaware of the disease[10].

The number of patients with POAG in 2010 is estimated to be around 4.5 billion in the world. At present 1.62 % to 3.5 % is the prevalence rate of POAG in the world [8].

Aravind Comprehensive Eye Study (ACES) estimated the prevalence of POAG to be 1.7 % in rural population of South India[11].

Prevalence rate of POAG between 40 and 50 years age group was 0.7 1%. About one fifth of the patients having POAG had blindness in at least one eye due to glaucoma.

Andhra Pradesh Eye Disease Survey (APEDS) estimated the prevalence of POAG to be 2.56 % and the Chennai Glaucoma Study (rural) CGS estimated it to 1.62 % and Chennai Glaucoma Study (urban) estimated it to 3.51 %. [10] But the Vellore Eye Survey (VES) estimated the least prevalence rate of POAG 0.41 % .[12]

PATHOGENESIS

Pathogenesis of glaucomatous damage in POAG :

It is postulated that the pathogenesis of POAG is multifactorial.

Two theories mechanical trauma theory and vascular perfusion alteration theory or a combination of the two proposed to explain the mechanism of POAG . Death of retinal ganglion cells occurs primarily by apoptosis (programmed cell death) and not by necrosis.

1. According to ischaemic theory , compromise of the blood flow in the microvascular structure resulting in ischaemia of the optic disc is responsible for that damage . The possible mechanisms include the following:

   - Loss of capillaries or loss of auto regulation of blood - flow in optic nerve head .

- Interference with the supply of nutrients to the axons and removal of metabolic waste products from axons .

2. Mechanical theory suggests that raised IOP causes direct damage to the retinal axonal fibres in their passage via lamina cribrosa. It is probably pressure gradient surrounding the optic nerve head producing that damage .

Once the initial injury occurs, a cascade of events results in astrocyte and glial cell mediation, as well as alterations in the extracellular matrix of the lamina cribrosa, leading to collapse of the cribriform plates and loss of axonal support.

Trabecular meshwork in eyes of POAG patients offers more resistance to the aqueous humour to pass-through. Aqueous humor out flow facility is a key determinant of IOP . Nearly 70–95% of the aqueous humor leaves the eye through the conventional outflow pathway, especially in older people . Higher resistance to aqueous outflow via the anterior chamber angle leads to increased IOP.

Histopathologic study of trabecular outflow system from eyes with POAG shows many abnormalities .

1. Fragmentation of collagen is seen and abnormal collagen like curly and long – spaced collagen are increased with more number of coiled bundle fibers .[23,27]
2. Thickening of basement membrane,
3. Inter trabecular spaces are narrowed . [24,28,29]
4. Fusion of beams in trabecular meshwork.[30]
5. Reduced endothelial cells in trabeculae[30]
6. Reduced number of actin filaments[31]
7. Accumulation of foreign material[25,26],
8. Giant vacuoles are reduced in number,
9. Narrowed collector channels[30],
10. Closing of Schlemm's canal[30].

Role of oxidative stress :

Recent evidences shows that reactive oxygen species (ROS) plays a major role in the pathogenesis of POAG . When compared to normal patients, the damage due to Oxidative DNA is more in the aqueous drainage system of glaucomatous eyes.[32] The role of ROS in the pathogenesis of glaucoma is reinforced by many experimental studies showing degeneration of trebecular meshwork made by hydrogen peroxide causes resistance to the flow of aqueous .In glaucoma the following pathways are altered . Pathways of Glutathione and Superoxide dismutase–catalase are altered. Increase in intraocular- pressure and visual – field defect severity in glaucomatous eyes corresponds to the severity of damage due to oxidative DNA which affects TM.[14] Oxidative stress, occurring in retinal ganglion cells, appears to play significant role in the cell death retinal neurons damaging the optic nerve head in glaucoma[33].

GENETICS

Recently, myocilin gene - MYOC (GLC1A) has been identified which causes juvenile – onset glaucoma and few cases (around 3–4%) of POAG in adult population.[34] Location of the causative gene is chromosome 1 q23–25 region.[35] About three

mutations in GLC1A gene is detected in four percentage of glaucoma patients.[36] Among three mutations, one specific mutation is responsible for major part of these abnormal genes seen in the Indian glaucoma population.[37] The mutation present in around 5% of the all glaucoma people . One more mutation is detected in a family from China having glaucoma of open-angle Juvenile type.[38] A new gene which is related with the onset of POAG in adults is placed in chromosome 2 (GLC1B) .[39]

RISK FACTORS FOR POAG :

Several ocular conditions are implicated as important risk factors related to glaucomatous damage of the optic nerve. These conditions include (1) increase in IOP, (2) old age, (3) positive history of having affected by glaucoma in the family members , (4) Latino or African ancestry, and (5) reduced central corneal thickness.

Risk factors also include myopia , reduced perfusion pressure in diastole , type II diabetic patients , and systemic hypertension .[1] Association of POAG with following factors like migraine, atherosclerosis , C reactive protein , body mass index, smoking are not proved .

AGE :

Old age is a major risk factor for POAG development.[39 -43] The impact of age in the prevalence of POAG persists after correcting for the association between increasing IOP and increasing age.[44] The prevalence in Japan is more in aged population where intra ocular tension never raises with age [45]. Age plays a major role in converting ocular hypertensive patients to POAG[46]. Many epidemiological studies proved that aging is a important risk factor in glaucoma development especially in population of African or Hispanic / Latino origin [47].

GENDER :

Most of the studies failed to prove the role of sex in the prevalence rate of glaucoma. Some studies showed that the glaucoma prevalence in high in men[48-51]. Barbados study stated that glaucoma was related with men in older age, increased IOP, low systemic BP, positive history of glaucoma in the family, low body mass and low intra ocular tension ratio.[52]

ETHNICITY / RACIAL FACTOR :

Ethnicity is an important risk factor for the development of POAG. Prevalence of POAG is high in black people than in white population [53]. They develop glaucoma at an young age then progress rapidly [43,54–57].

It has been estimated that in United States, the prevalence and incidence of blindness of eye due to POAG is eight to ten times high in black people with POAG than in white people with POAG[43,58].

Open angle glaucoma of primary type is comparatively less in Pacific Island people[59], some part of Asian people[60–62], and some native tribes of north America. The prevalence and incidence of POAG is more in West African people , Afro – Caribbean people, or Hispano / Latin people when compared with other ethnic groups [9,43]. POAG prevalence is three times more in Hispanics of Mexican origin[9]

FAMILY HISTORY:

Positive history of family members having glaucoma is proven risk factors for POAG. About five to fifty percent cases of glaucoma of open angle primary type have hereditary origin, with the estimation of twenty to twenty five percentage [63]. The risk of getting glaucoma in relatives of first degree is 4–16%.[63] Since only ten to sixty percent of patients where glaucoma is diagnosed, a negative family history of glaucoma could be not accurate.

" When all relatives of first degree of patients detected to have glaucoma of open angle primary type from Rotterdam study examined 22.4 percent of them were found to have glaucoma. This is nearly 10 times greater than the risk in the general population". A population based twin study calculated the risk of inheritance of POAG to be around 13%.[64] Barbados study showed that one quarter of the siblings of POAG patients had POAG or glaucoma suspect.[65] Siblings have high possibility of raised IOP and a larger cup – to – disc ratio .[66,67]

MYOPIA :

Many of the epidemiologic cross – sectional studies done in Asian Indians , Hispanicians , Chinese, Whites , Afro – Caribbeans , then Japan people shows that myopic patients have a high risk of developing POAG when compared with normal patients.[68-74]

LALES has given the evidence of association between increased axial length (axial myopia) and a increased POAG prevalence.[75] The hypothesis is that weaker sclera in myopic eyes gives in adequate support for the optic nerve, resulting in a greater susceptibility for the optic disc to glaucomatous changes .

INTRA OCULAR PRESSURE :

It is a well established risk factor in the pathogenesis of glaucoma of open angle primary type. There is clear evidence indicating increased IOP causes glaucomatous changes optic nerve head in experimental study animals.[76, 77] The OHTS study shows that risk of developing POAG is up to six times higher in ocular hypertension than in those without any risk factors for glaucoma.[46]

The risk of developing POAG in ocular hypertension has been estimated to be 1–2% per year or about 10% per decade. Recent data from the Ocular Hypertension Treatment Study indicates that this risk could be higher at 9.5% over five years.[46] The risk of developing glaucoma in ocular hypertension is suggested to rise with IOP, with risk significantly increased for a baseline IOP of 24 mm Hg or greater, and especially for IOP over 30 mm Hg.

CENTRAL CORNEAL THICKNESS :

The mean CCT of normal eyes is not a constant value. It differs in various races or ethnic population .The CCT in Caucasians is 0.556 mm, in Latinos 0.546 mm and in African Americans it is 0.534mm.[78] Many studies done across multiple ethnicities showed an increase in prevalence and incidence of glaucoma as IOP increases.

A thinner central cornea is an independent risk factor in the transformation of ocular hypertensives to glaucoma of open angle primary type.[79] Standard nomogram have been published, for correcting applanation IOP measurements for CCT.[80] Studies showed that the corneal biomechanics differs among population and have a significant impact on errors in measurement of intraocular

pressure than CCT . So, thinner corneas could be a marker in the higher susceptibility of the optic disc .

TYPE 2 DIABETES MELLITUS:

Even though the initial studies showed weak or no association of diabetes mellitus type II to glaucoma of open angle primary type,[95-103] support obtained from the population – based and cross-sectional or cohort studies shows that diabetes mellitus is a potential risk factor in the pathogenesis of glaucoma of open angle primary type.[95-97,100,102] Population –based study of "Hispanics (in Los Angeles, California),[96] non–Hispanic Whites (in Beaver Dam, Wisconsin and Blue Mountains, Australia), [95,102] and a large cohort enrolled in the Nurses' Health Study[100]" all implied that patients of DM type II have high risk (40% higher odds in Hispanics, two fold higher odds in non-Hispanic Whites) to develop glaucoma of open angle primary type.

LALES,[96] states that more the duration of DM , more the risk of developing POAG. Micro vascular changes occurring in the optic head in diabetic patients can contribute to the higher chances of glaucomatous damage to POAG .[101]

OCULAR PERFUSION PRESSURE :

Ocular perfusion pressure is defined as the mean arterial pressure (MAP, at eye level) minus the intra ocular pressure. Mean ocular perfusion pressure is calculated as two-thirds of arterial pressure minus IOP. Large population studies have determined that reduced diastolic perfusion pressure is related with more risk of developing glaucoma.

Barbados eye study showed that individuals with lowest diastolic perfusion pressure have the 3.3 % risk of developing glaucoma. It is proved that low ocular perfusion pressures alters blood circulation at the surface of the optic disc and leads to glaucomatous damage of the optic disc [81].

Early Manifest Glaucoma Treatment Trial states that lower systolic perfusion pressure is associated with increased risk of worsening of glaucoma (relative risk is 1.42) during the period of eight years.[82]

OTHER RISK FACTORS :

Migraine and disorders of peripheral vasospasm are also detected to have a part in the pathogenesis of glaucoma of open angle primary type. Other factors like co-existing systemic hypertension and cardiovascular system diseases are not found to have the role consistently.

INVESTIGATIONS

1. NON - CONTACT TONOMETRY

"Principle : Measuring the time it takes from the initial generation of the puff of air to where the cornea is exactly flattened in milliseconds to the point where the timing device stops gives an indirect measurement of IOP". [90-92]

Noncontact tonometer have generally been considered a fast and simple way to screen for high IOP. Some tonometers have two sensors. The first is for light and the second is for pressure. Cornea is applanated by air puff. The light sensor catches the applanation moment, and the pressure sensor watches the chamber to record the intraocular tension. Measurement range is 0 to 60 mmHg.

Advantages :

1. Measurement takes relatively very less time.
2. Easy to do the procedure.
3. Topical anaesthetic eye drops are not needed.
4. Trained para-medicals can do the procedure.
5. Useful for the mass screening programmes.
6. No risk of transmission of infections.
7. Good co-operation from the patient as there is no contact with the eye.

Figure 1 : Technique of NCT

Figure 2 : Calibration of NCT

1. APPLANATION TONOMETRY

Principle:

It is based on Imbert - Fick law : An external force (F) against a spherical object is equal to the pressure in the sphere (P) multiplied by the area flattened by the external force (A) . $F = P \times A$.

Preparation :

Variety of techniques are described for disinfecting tonometer tips: Soaking applanation tip for 5 to 10 minutes in diluted sodium hypochlorite, 3% H_2O_2 or 70% iso propyl alcohol or by wiping with povidone iodine. Ten minutes of rinsing in tap water and disposable cover for tips can also be used for sterilisation. Always clean the tip with sterile cotton swab after sterilisation to clean any residues if present.

Method :

Patient is made to sit comfortably in front of the slit lamp: Head position should be correct, forehead should touch the head band and the chin touching the chin rest .

Instil the topical anaesthetic eye drops 4 % xylocaine or paracaine is used. Conjunctival cul-de sac is stained with sodium fluorescein.

1. Fluorescein facilitates visualization of tear meniscus at the margin of contact.

2. The cornea and the biprism are illuminated with cobalt blue light . Light source from the slit is shown from right side of the patient for his right and from left side for the examination of his left eye.

3. Patient should look straight ahead, gently separate the lids and the slit lamp is slowly moved forward so that the tonometer tip touches the cornea.

4. Fluorescent semi - circles are viewed through the biprism.

5. Force against the cornea is adjusted until the inner edges of the semicircles overlap.

6. Ocular pulsations create excursions of semi-circular tear meniscus and IOP is read as the median over which arc glides. This is the end point at which a reading can be taken from a graduated dial which indicates grams of force applied to tonometer and so this number is multiplied by 10 to obtain IOP in mmHg.

7. Then the same procedure done for the other eye.

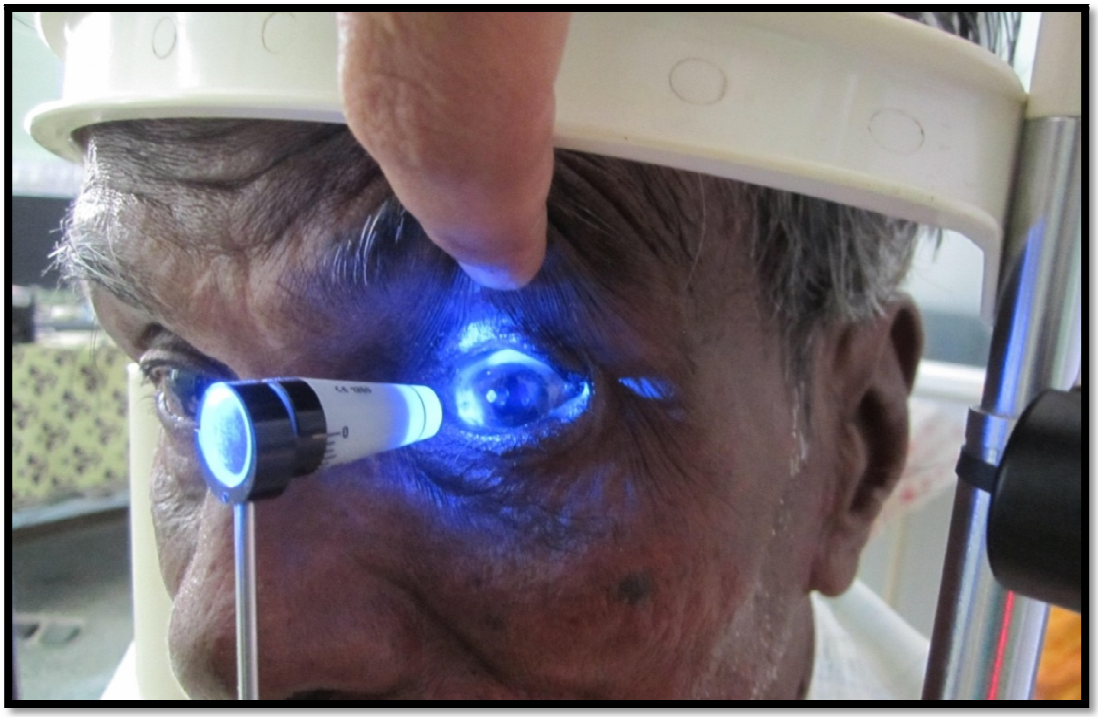


Figure - 3 Technique of Applanation Tonometry

ULTRASOUND PACHYMETRY :

It is a contact technique with no need for coupling fluid . The piezo electric crystal produces ultrasound waves . It is delivered by a probe tip . Time needed for the ultra sound waves to travel through the cornea and the velocity of ultra sound waves through the cornea are calculated and CCT is calculated . The ultra sound frequency used is 10 to 20 MHz. This technique is most commonly all over the world. It is a simple procedure to perform and fast also . Ultrasound pachymeters are easily portable, highly accurate and very reliable . Very low corneal thickness like 125 microns can also be measured . It has got an inbuilt algorithm to adjust for the IOP calculated by appplanation according to the CCT. It is provided with a printer and it enables a easy documentation. But this technique may underestimate CCT in edematous corneas.

The pachymetry device used was calibrated so that the mean CCT was 545 µm. Values lesser than this, may underestimate the IOP and values higher than this may overestimate the IOP. So corrected IOP was calculated for all the patients.

Figure – 4 Technique of Pachymetry

GONIOSCOPY :

Gonioscopy is " biomicroscopic examination of the anterior chamber angle structures of the eye, where aqueous humor gains access to Schlemm's canal". Normally, the angle of the anterior chamber cannot be viewed during slit lamp examination as the light rays emanating from the angle strike the cornea at an angle steeper than the critical angle 46^{0} and are refracted back to the eye due to total internal refraction. The solution to this problem is to eliminate the cornea optically. This can be done by using gonioscopy lenses which are designed to overcome the total internal reflection and abolishes the critical angle by altering the cornea-air-fluid interface.

Procedure :

a. The patient should be explained about the procedure so that the patient will be aware of the contact of the gonioscope with the eye patient is advised to keep the eyes open during the procedure and not to change the position of the head.

b. Slit lamp illumination should be minimum with thin section of the slit to avoid miosis. Background illumination should also be reduced.

c. Topical anaesthetic solution is instilled 4 % xylocaine or paracaine eye drops are used.

d. Coupling fluid is needed when Goldmann single mirror is used.

e. Inserting the goniolens is made easier by asking the patient to look upwards. Then gently insert the lens with its leading upper edge holding the upper lid margin to prevent any reflex blink. Then the patient should look directly ahead.

f. Initially the inferior angle is visualised first by rotating the gonioscope so that the mirror is at 12 0 ' clock position and then clockwise rotation is done to view rest of the angle areas

g. In eyes with convex iris pattern , 'over the hill ' view is done by requesting to look in the mirror direction .

h. In cases where iris plane is flat , the patient is requested to look away from the mirror to get a view parallel to the iris with good quality image .

Figure 5 : Gonioscopy

Figure 6 : Angle structures in Gonioscopy

Automated perimetry

The central 30^0 visual field examination using automated perimetry is currently the gold standard in evaluation, management and follow – up of POAG .[1] Bebie Fankhauser, Hirsbrunner contributed the concepts for the development of Octopus perimetry.

Octopus perimetry has got the normal full threshold testing strategy, the dynamic strategy (Weber) and the Tendency Oriented Perimetry (TOP).

The seven in one report is interpreted in the following order :

1. Patient data includes the demographic data of the patient .
2. Examination data highlights the examined eye , size of the pupil , examination programme , strategy and reliability .
3. Value table shows actual measured values of local sensitivities in at each of the test locations .
4. Comparison table represents the local difference between the measured values and the normal values valid for the patients age.

5. Corrected comparison table shows the defects discounting any uniform depression caused by a cataract or other diffuse loss.

6. Gray scale is an overview of the pattern of visual field defects for the doctor and can be used to explain the nature of the problem to the patient .

7. Cumulative defect (Bebie) curve is an arrangement of all the test data from the highest value to least from left to right which is overlaid on a statistically age corrected normal for comparison.

8. Probability plots gives the graphical representation of the probability or significance of a defect.

9. Visual field indices condenses the visual field results in a few numbers.

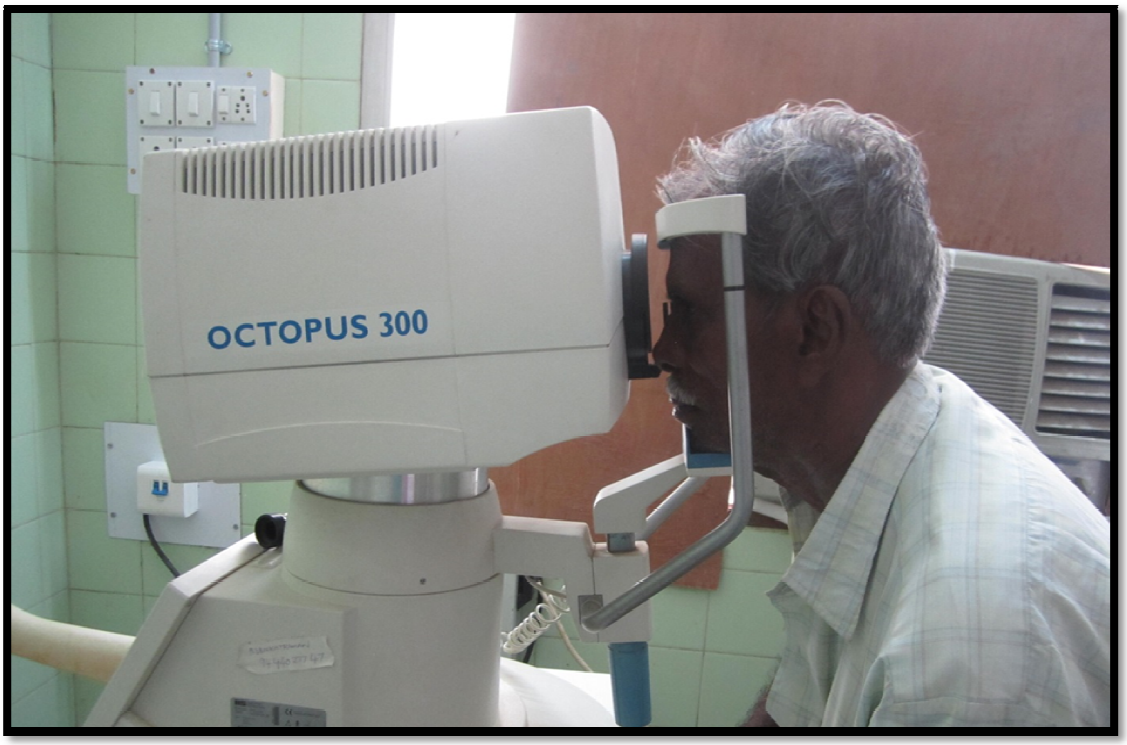


Figure 7 : Technique of Octopus Automated Perimetry

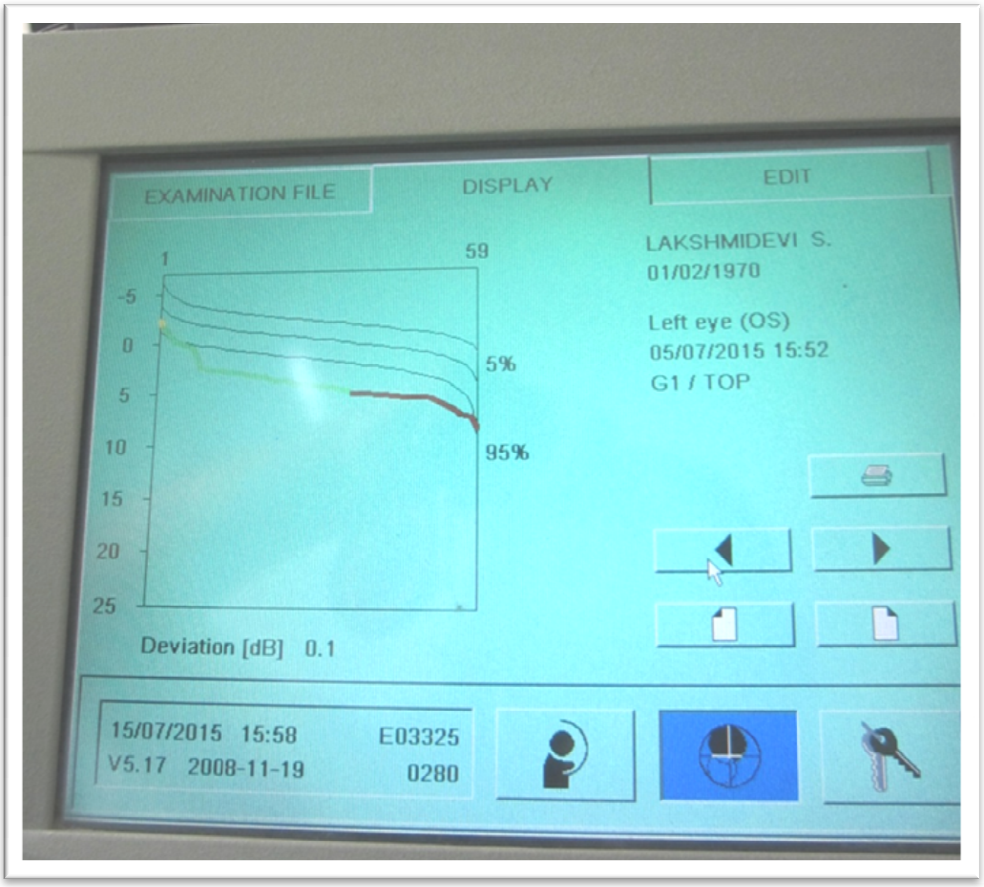


Figure 8 : Bebie's curve in Octopus Automated Perimetry

Optic Nerve Head Evaluation Techniques

The overlap seen between normal and those with glaucoma is high. A thorough clinical examination of the optic nerve head to quantify the structure of the optic nerve and surrounding retina is essential to distinguish between the two.

The purpose of ONH evaluation is :

- To assess the health of ganglion cell axons and to distinguish between the healthy and the diseased ONH.
- Grouping the patients into categories such as healthy, mild, moderate, and advanced glaucoma.
- Monitoring change / progression.
- Quantifying the rate of any change that has occurred.

Ideally the examination should be under magnification and a stereoscopic view.

Direct Ophthalmoscope

The use of parallax and attention to the bending of vessels after they cross the disc margin provides a reasonably fair idea about the contour of the rim.

Figure 9 : Technique of Direct Ophthalmoscopy

Figure 10 : Glaucomatous fundus

Figure 11 : Glaucomatous fundus

DIABETES MELLITUS

" Diabetes mellitus is defined as a group of metabolic disorders characterized by chronic hyperglycemia associated with disturbances of carbohydrate, protein and fat metabolism due to absolute or relative deficiency in insulin action and or secretion".

As diabetes mellitus is a chronic disease with no absolute cure , it causes damage, dysfunction and ultimately failure of various organs in our body chiefly blood vessels, eyes, heart, kidneys and nervous system .

International Diabetes Federation, in 2013 had calculated the prevalence of diabetes in the world and the estimated population having diabetes was 381 million.[86] Its incidence is alarmingly increasing, and in 2030, the estimated prevalence is going to double.[87] It calculated the number of patients with diabetes mellitus in India and the number is 40.9 million and the value is going to rise up to 69.9 million by 2025 .

The mean age of onset of diabetes type II is 42.5 years.[88] The higher incidence rate is related to genetic susceptibility and life style

changes like intake of high – calorie diet, sedentary lifestyle in India's ever growing medium socio economic class.[89]

REVIEW OF LITERATURE

Bonomi L *et al.* (2000) [93] stated that glaucoma of open angle primary type is more seen in eyes having low perfusion pressure . The total number of diabetic patients are high in group I (patients with low perfusion pressure) cases are 22.4% compared to group II (patients with normal perfusion pressure) are 12.7% and the presence of significant difference confirms that diabetes is one of the risk factor to develop glaucoma of open angle primary type .

Pasquale LR *et al.*[94] in his prospective study of diabetes mellitus Type II and the risk of primary open angle glaucoma in women had shown a positive relationship between Diabetes Mellitus type II and POAG.

James M . Tielsch *et al.*[43] in Baltimore eye survey done on American and African American population with diabetes mellitus had 5308 participants. POAG was diagnosed in 161 patients. When compared with the non diabetic population there was no increased incidence of POAG in diabetic patients.

Paul Mitschel *et al.*[95] had explored "the relationship between diabetes and open - angle glaucoma in a defined older Australian population in Blue Mountains Eye Study". Three thousand six hundred fifty – four people of between 49 and 96 years old, undergone a complete eye examination to diagnose POAG. They found out that Glaucoma prevalence was increased in patients with diabetes mellitus (5.5 %), when compared with the patients not having diabetes (2.8 %). 13 % of patients with glaucoma had type II diabetes, and diabetes type II was present in 6.9 % of patients without POAG. He concluded that 'significant and consistent relationship between type II diabetes mellitus and POAG was present, which appeared to be non dependent on the effect of diabetes on intra ocular tension' , suggesting that there is a definite association between them .

Vikash Chopra *et al*[15] in his population – based cross - sectional study, examined the "association between type 2 diabetes mellitus (T2 DM) and the risk of having open – angle glaucoma (POAG) in an adult Latino population" . The study concluded that the type II DM occurrence and a years of type II DM were independently associated with a high risk of developing POAG in the LALES cohort .

Simone de voogd *et al*[101] did a population - based prospective cohort study to investigate diabetes mellitus related as high risk for glaucoma of open angle primary type . This Rotterdam study was done in Netherlands and the follow – up period was three years . This study clearly proved that the diabetic patients did not have any increased risk of developing POAG .

M. Christina Leski *et al*[65] evaluated " the risk factors for definite Primary open – angle glaucoma (OAG), based on African - descent participants of the Barbados Eye Studies". It was a cohort study having a follow – up period of nine years. This was a early estimation for risk factors of glaucoma of open angle primary type incidence long duration. The study is again based on a good volume of newly diagnosed patients.

Incidence was found to be higher in the African origin patients, where the factors like old age, increased intra ocular tension, and positive family history contributed to the risks. Other predictors were low diastolic BP and low ocular perfusion pressure, which doubled the risk. Thin CCT was found to be also have higher risk. The findings showed a multi factorial pathogenesis of glaucoma and advises that risk factors can also be applied across various populations .

LR Pasquale *et al*[100] in his prospective cohort study analysed the association between "diabetes mellitus type II and the incidence of primary open angle glaucoma in women as a part of Nurse's Health Study". The outcome measures are multivariaiable rate ratios of POAG and assoiciated 95 % confidence intervals derived from proportional models. They concluded that presence of diabetes mellitus type II is a definite risk factor for glaucoma of open angle primary type in women.

S. Bonovas *et al*[97], in his meta - analysis of various studies which were published in many highly acclaimed peer - reviewed journals found out that the risk of POAG in patients with diabetes is statistically significant.

John D Ellis *et al* [46] has done a cohort study for evaluation of diabetes as a major risk factor in glaucoma of open angle primary type or ocular hypertension . Study people composed of 6631 diabetic patients and 166144 non – diabetic people aged more than 40 years and followed up for 24 months period . The study could not confirm the relation between ocular hypertension and POAG.

Nakamura *et al*[103] in his experimental study found out that diabetes mellitus damages vascular tissues and also causes compromise of retinal glial neuronal functions . It affects the

metabolism of the anterior layers of the retina . All these disturbances lead to apoptosis of retinal ganglion cells . It also renders retinal ganglion cells more susceptible to the effects of raised intrao cular pressure. Retinal samples from rats eye with diabetes caused by Streptozotocin were taken. He cauterized the episcleral veins to make the eye chronic glaucoma, had high number of apoptotic cells.

AIM OF THE STUDY

AIM :

Aim of the study is early detection of development of Primary Open Angle Glaucoma in patients having diabetes mellitus. Early detection can avoid irreversible optic nerve damage caused by Primary Open Angle Glaucoma.

MATERIALS & METHODS

DESIGN OF THE STUDY :

This was a cross sectional hospital based study of diabetics attending Diabetology OPD, Coimbatore Medical College Hospital, Coimbatore for early diagnosis of Primary Open Angle Glaucoma.

STUDY SETTING :

Study was done at Department of Ophthalmology, Coimbatore Medical College Hospital, Coimbatore .

STUDY PERIOD :

This study period was about 12 months extending from August 2014 to July 2015.

STUDY POPULATION :

The study was done on patients attending the Diabetology OPD based on selection criteria . A minimum of 200 patients were included in the study.

Before commencing the study Ethics committee approval was obtained from the Coimbatore Medical College and Government Hospital .

Diabetic patients attending Diabetology OPD were screened for POAG after obtaining consent .

INCLUSION CRITERIA :

1. Patients without any visual complaints .
2. Patients of age above 40 years .
3. Both sexes .
4. Normal anterior and posterior segment examination of both eyes.

EXCLUSION CRITERIA :

1. Already diagnosed glaucoma patients .
2. Family history of glaucoma .
3. Associated ocular pathology .
4. Associated systemic diseases like Hypertension and Thyroid Eye Disease.
5. Patients with H/O smoking and alcohol consumption .
6. Pregnancy.
7. Fundus showing diabetic retinopathy changes .
8. Optic nerve head anomalies.

9. History of ocular trauma.

STUDY METHODS :

All the patients aged 40 years and above attending Diabetology OPD were selected on the basis of the above criteria. The sampling technique adopted for this study was non – probability purpose sampling technique. All the patients were explained about the study and written consent was obtained. Data collected using structured questionnaire which comprises socio demographic characteristics like age, sex, occupation & detailed history. Then ophthalmic examination was done.

Ophthalmic examination included

1. Uncorrected visual acuity was measured with Snellen chart. Refraction was done and the best corrected visual acuity was noted.
2. Intraocular pressure (IOP) measurement was done with Non contact tonometer. (NCT 200)
3. Detailed anterior segment examination using slit lamp was done to rule out other types of glaucoma and associated ocular pathology.
4. Detailed fundus examination under full mydriasis obtained by 0.8 % tropicamide and 5 % phenylephrine was done with Direct ophthalmoscopy, 90D and Indirect ophthalmoscopy.

In all patients suspected of having signs of Primary Open Angle Glaucoma , the following tests were done for both the eyes.

1. Applanation tonometry was done with Goldmann 's applanation tonometer.
2. Corrected IOP was calculated after measuring CCT by ultrasonic pachymetry (PAC SCAN 300 P).

3. Peripheral anterior chamber angle study was done with Goldmann' s single mirror Gonioscopy.

4. Fundus photo was taken for documentation and

5. Perimetry was done with Octopus automated perimetry. (OCTOPUS 300).

The data analysis and interpretation was done using SPSS 16 version. Descriptive and inferential statistics was adopted.

STATISTICAL ANALYSIS

Total number of patients included in the study : 200

TABLE – 1

Age distribution

Age	**Number of patients**	**Percentage**
40 – 50 years	138	69 %
5I – 60 years	62	31 %
Total	200	100 %

CHART– 1

Age distribution

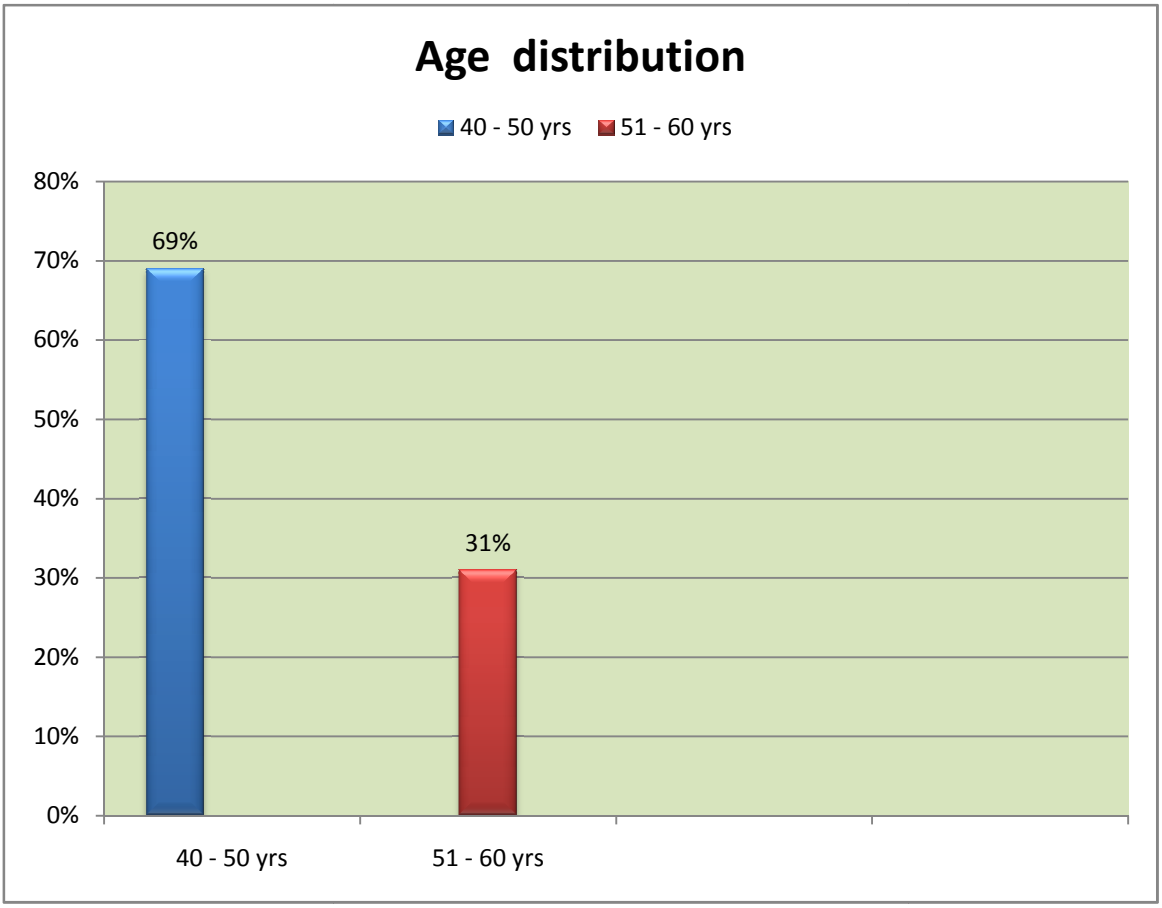


Table – 2

Gender distribution

Gender	Number of patients	Percentage
Male	119	59.5 %
Female	81	40.5 %
Total	200	100 %

CHART – 2

Gender distribution

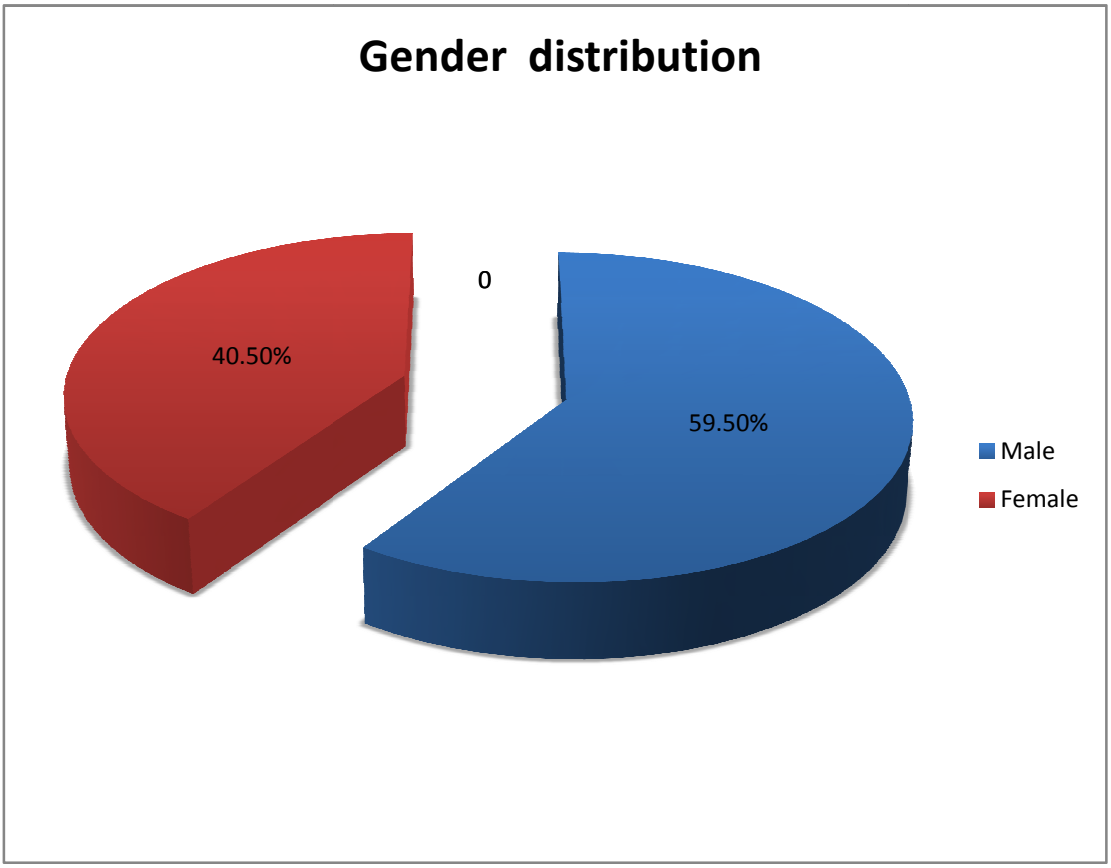


TABLE – 3

Duration of DM

Age	Number of patients	Percentage
Less than 5 years	138	69 %
5 – 10 years	60	30 %
Above 10 years	2	1 %
Total	200	100 %

CHART – 3

Duration of DM

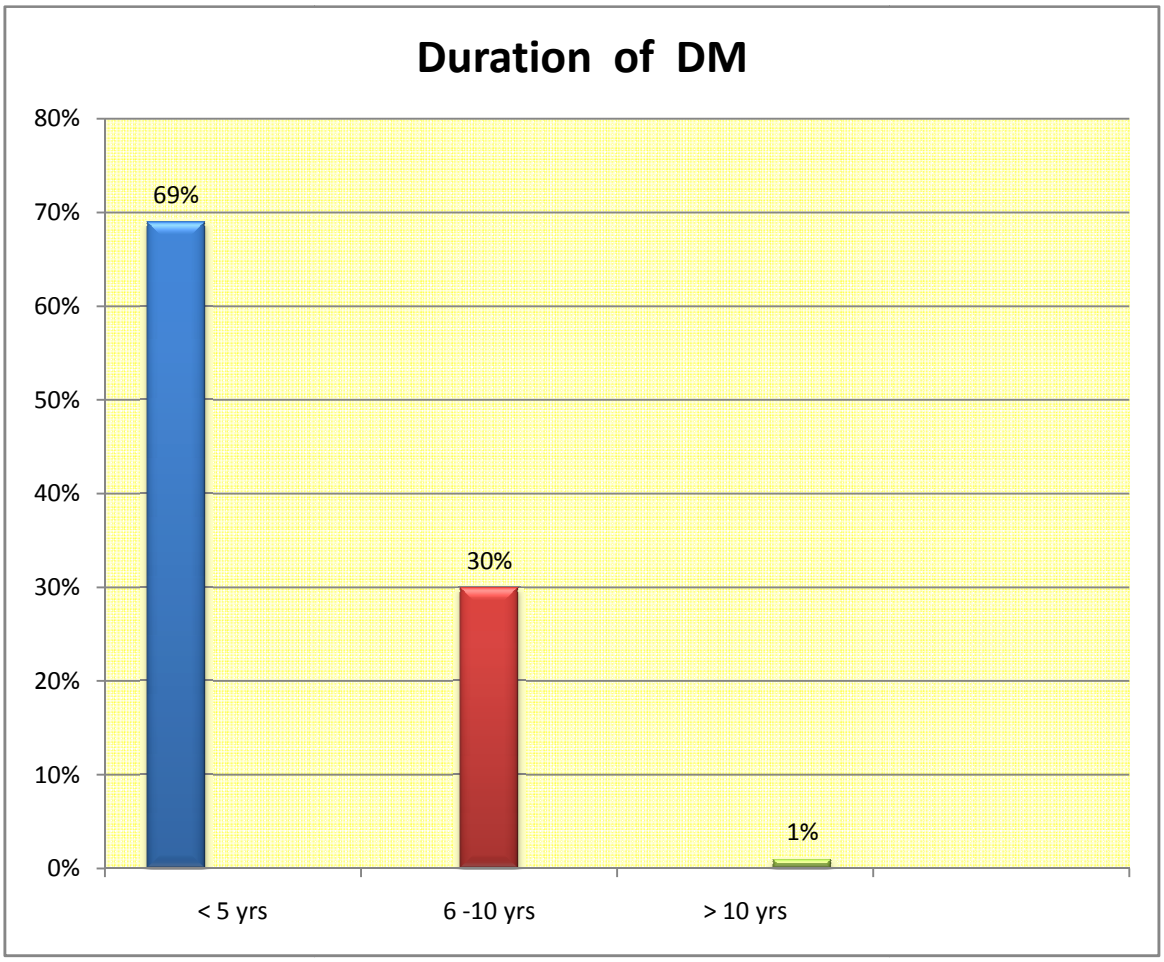


TABLE - 4

Type of DM

Type of DM	Number of patients	Percentage
Type I	4	2 %
Type II	196	98 %
Total	200	100 %

CHART- 4

Type of Diabetes Mellitus

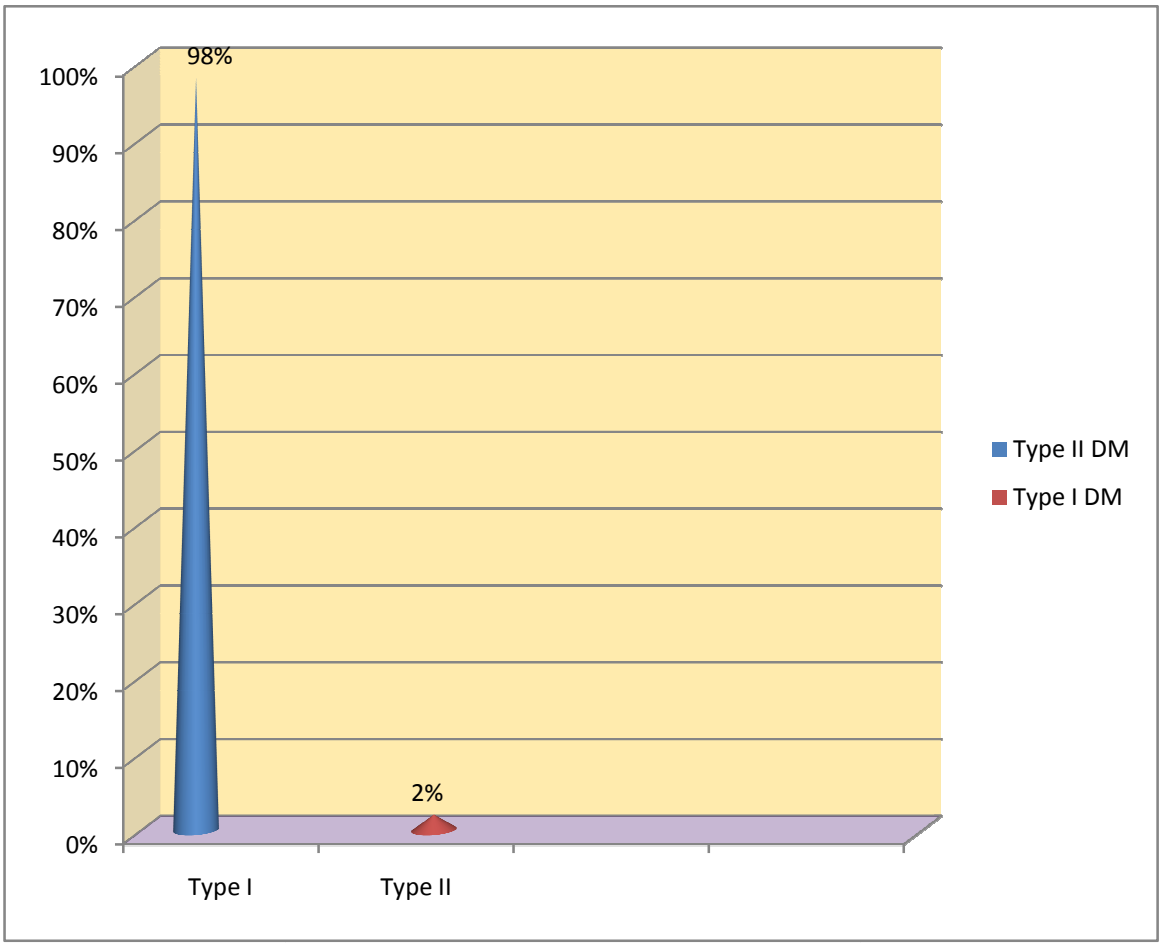


TABLE – 5

Fundus Examination

Fundus Findings	Number of patients	Percentage
Normal optic disc	191	95.5 %
Suspicious optic disc	*9	4.5 %
Total	200	100 %

* Patients with IOP > 20 mm Hg by NCT

CHART – 5

Fundus Examination

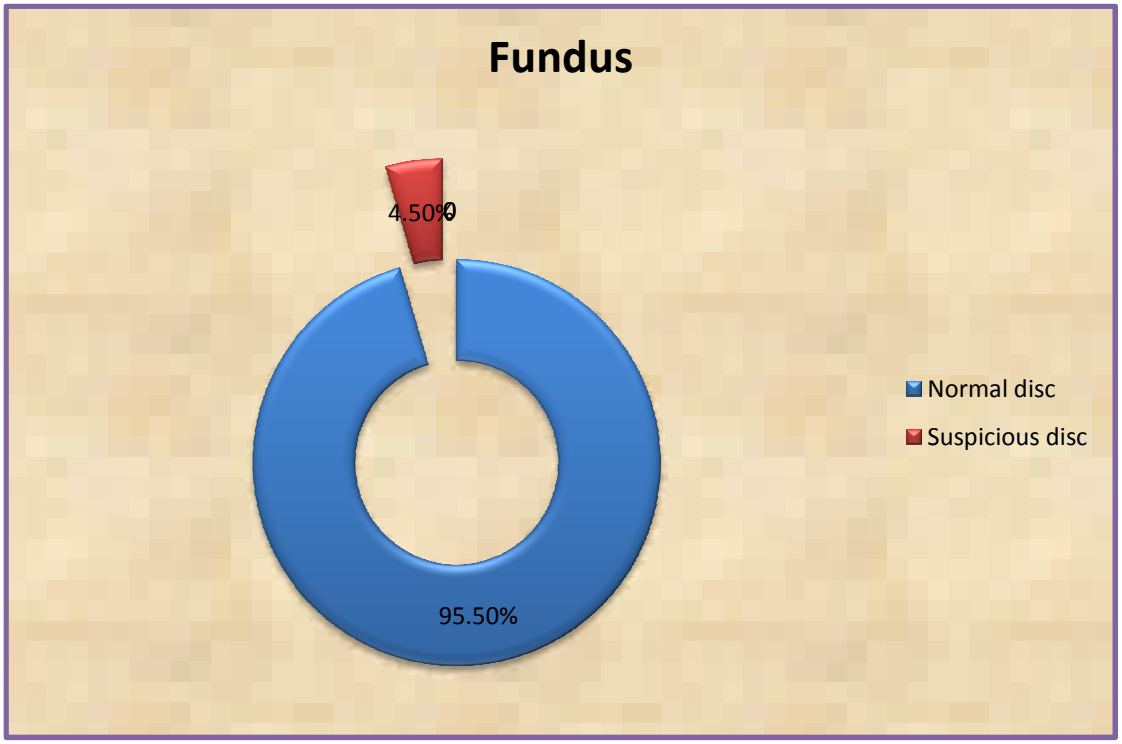


TABLE -6

Non Contact Tonometry

NCT	Number of patients	Percentage
Normal (≤ 20 mm Hg)	186	93 %
High (> 20 mm Hg)	14	7 %
Total	200	100 %

CHART -6

Non Contact Tonometry

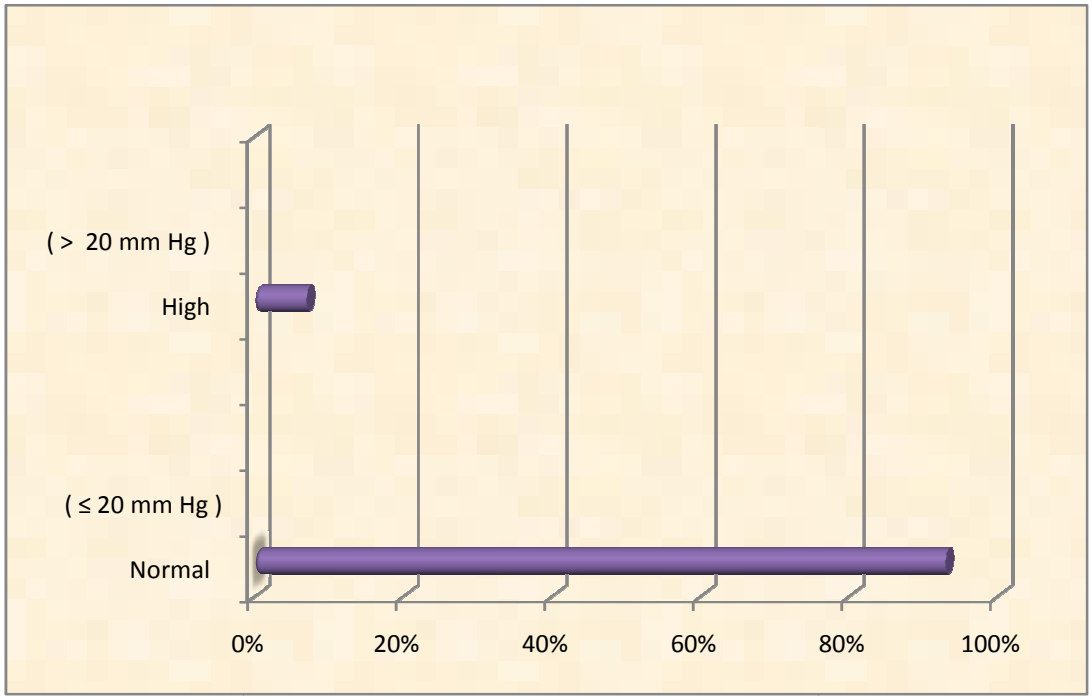


TABLE – 7

Central Corneal Thickness values

in patients with IOP > 20 mm Hg by NCT

CCT	Number of patients	Percentage
540 – 550 µm	2	14.3 %
551 – 560 µm	6	42.9 %
561 – 570 µm	3	21.4 %
571 – 580 µm	3	21.4 %
Total	14	100 %

CHART – 7

Central Corneal Thickness values

in patients with IOP > 20 mm Hg by NCT

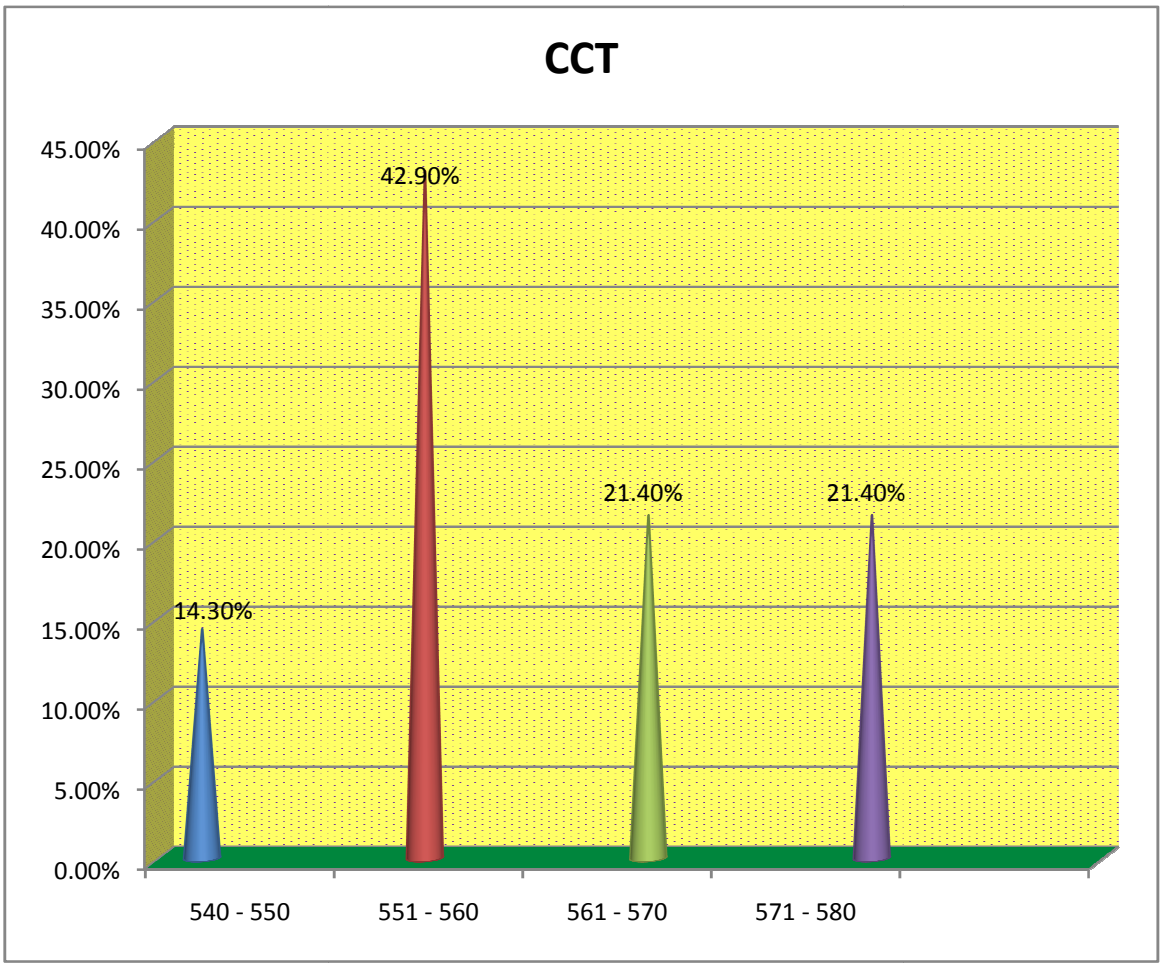


TABLE – 8

Corrected Intra Ocular Pressure values

in patients with IOP >20 mm Hg by NCT

Corrected IOP	Number of patients	Percentage
High (> 20 mm Hg)	9	64.3%
Normal (≤ 20 mm Hg)	5	35.7%
Total	14	100 %

CHART – 8

Corrected Intra Ocular Pressure values

in patients with IOP >20 mm Hg by NCT

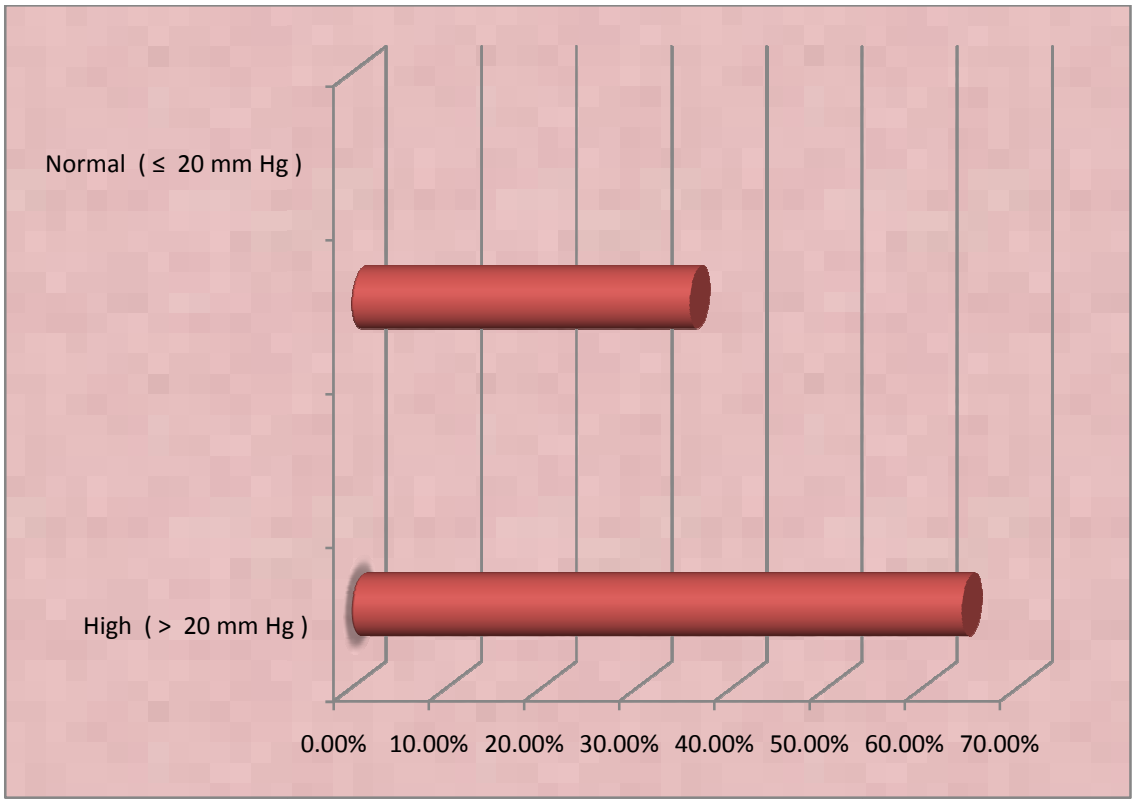


TABLE – 9

Automated Perimetry tracing

in patients with IOP > 20 mm Hg by NCT

Automated Perimetry	Number of patients	Percentage
Depression	9	64.3 %
Normal	5	35.7%
Total	14	100%

CHART – 9

Automated Perimetry tracing

in patients with IOP > 20 mm Hg by NCT

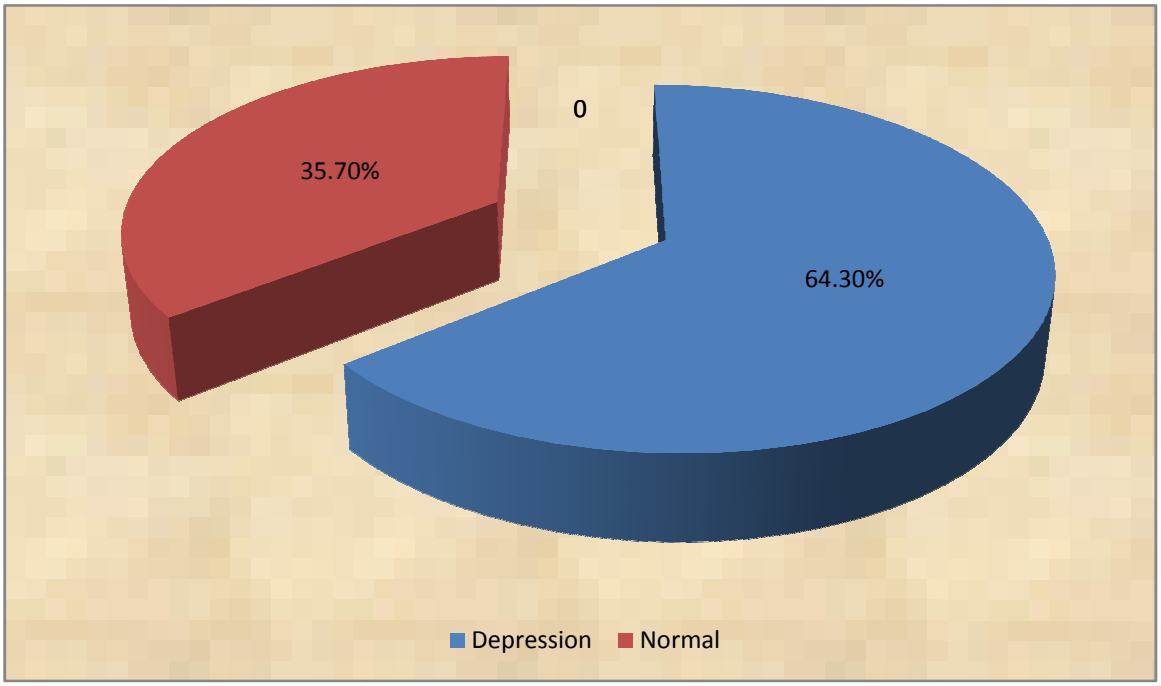


TABLE – 10

Corrected IOP (> 20 mm Hg) with CCT

No of patients with Corrected IOP > 20 mm Hg	CCT (μm)			
	540 – 550	551 – 560	561 - 570	Total
	2	6	1	9
Percentage	22.2 %	66.7 %	11.1%	100.0%

CHART – 10

Corrected IOP (> 20 mm Hg) with CCT

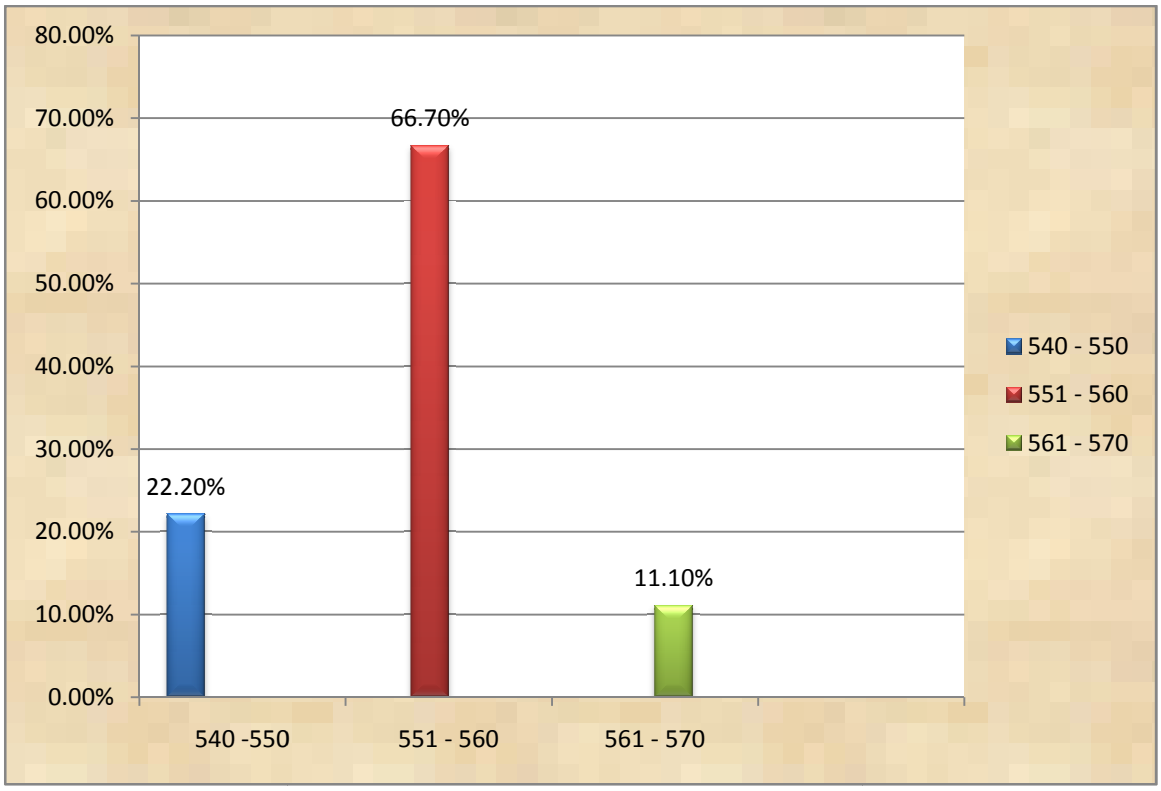


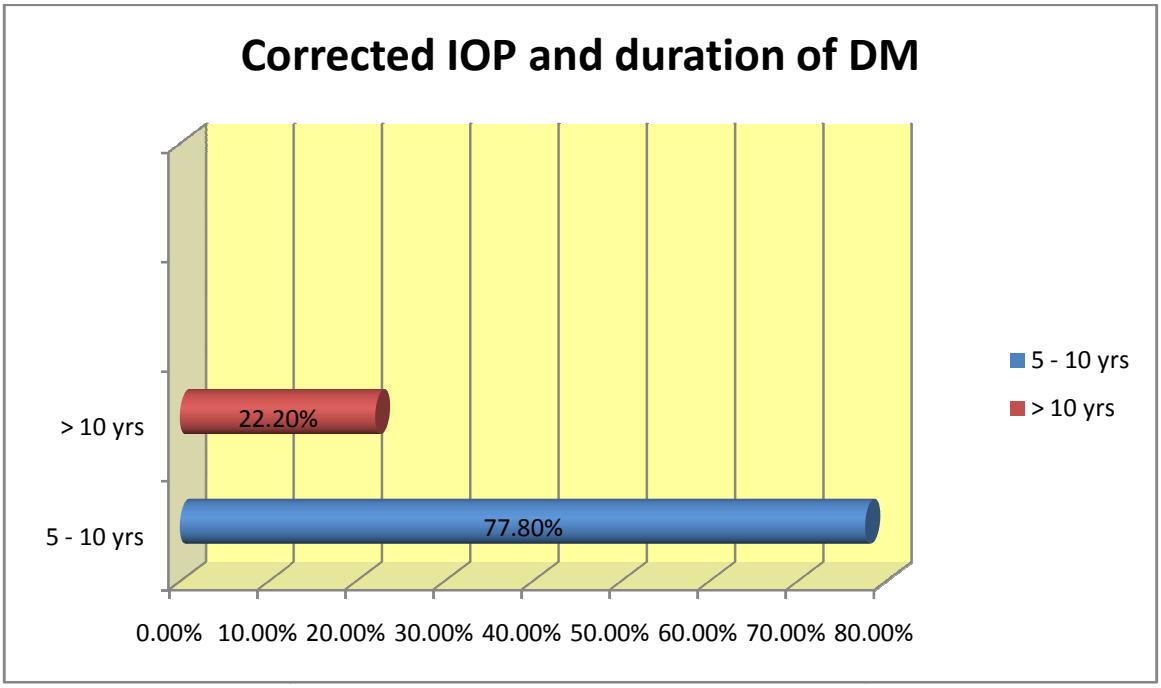
TABLE – 11

Corrected IOP (> 20 mm Hg) with duration of DM

Duration	Number of patients	Percentage
5 – 10 years	7	77.8 %
Above 10 years	2	22.2 %
Total	9	100 %

CHART – 11

Corrected IOP (> 20 mm Hg) with duration of DM



DISCUSSION

Our country has got one of the highest incidences of Diabetes of both the types - I & II. As diabetes causes damage of the vascular system of various organs in the body, complications are more and life – long follow - up is needed.

Eyes are also commonly affected in diabetes as it causes micro angiopathy of retinal vessels. It acts as an important risk factor in the pathogenetic process of glaucoma of open angle primary type. Glaucoma with open angles primary type does not cause any symptoms . When it reaches advanced stage, it causes gross reduction in field of vision and loss of vision, which cannot be reversed.

So early diagnosis of glaucoma is of para-mount importance.

As diabetes and glaucoma of open angle primary type have polygenic inheritance , it is necessary to screen all the diabetic patients for glaucoma . Hence all diabetic patients were screened for open angle glaucoma of primary type . In our present study , a total of 200 patients with diabetes mellitus were examined for glaucoma of open angles primary type .

Among the total of 200 patients, 138 patients were in the 40 – 50 years age group (69 %) , and the remaining 62 patients were in the age group of 51 – 60 years (31 %) . Patients above the age of 60 are not included as chances of having cataract are more.

Gender distribution showed no significant difference between men and women. 119 patients were men (59.5 %) and 81 (40.5 %) patients were women .

Among 200 diabetic patients, Type II diabetes mellitus was present in 196 patients (98 %) and Type I diabetes mellitus was present in 4 patients (2%).

When patients are group for duration of diabetes, 69 % (138 patients) were in < 5 years group. 5 – 10 years group comprises of 60 patients forming 30 % . Above 10 years duration group comprises only 2 patients forming 1 % .

Fundus examination showed normal optic disc in 191 patients (95.5 %) and suspicious optic disc in 9 patients (4.5 %) .

IOP measurement with NCT and Goldmann 's Applanation Tonometry were done in the same time of the day (morning) as normal diurnal variation of IOP may interfere with correlation. Non contact tonometry showed normal IOP (≤ 20 mm Hg) in 186 patients (93 %) and high IOP (> 20 mm Hg) in 14 Patients (7 %) .

Goldmann 's Applanation Tonometry was done on these 14 patients, showed high IOP (> 20 mm Hg) .

Goldmann 's single mirror gonioscopy was done on these 14 patients, showed open angles .

In our study , the pachymetry device used was calibrated in such a way that the mean CCT was 545 µm. Any value below this, will underestimate the IOP and any value above this will overestimate the IOP. So corrected IOP was calculated for all fourteen patients .

CCT done for 14 patients were divided in to five groups . 540 – 550 µm group comprised two patients (14.3 %), 551 – 560 µm group six patients (42.9 %), 561 – 570 µm group three patients (21.4 %) , 571 – 580 µm group three patients (21.4 %) .

Corrected IOP were calculated for 14 patients, nine patients had IOP > 20 mm Hg and five patients had IOP ≤ 20 mm Hg .

CCT was analysed and grouped for 9 patients with high corrected IOP > 20 mm Hg , 66. 7 % belongs to 551 – 560 µm group , 22.2 % belongs to 540 – 550 µm group , and 11.1 % belongs to 561 – 570 µm group .

Automated perimetry tracing showed , nine patients (64.3 %) had depression and five patients (35.7 %) had normal perimetry.

Goldmann Applanation Tonometry was correlated with the duration of diabetes. 85.7 % of diabetic patients belong to 5 – 10 years duration group and 14.3 % of diabetic patients belong to > 10 years duration group.

Corrected IOP was correlated with the duration of diabetes. 77.8% of patients had 5 – 10 years of diabetes and 22.2 % of patients had diabetes > 10 years.

SUMMARY

- Among 200 patients evaluated, IOP was elevated in 14 patients when measured with NCT .
- Out of 14 patients corrected IOP (Goldmann 's Applanation Tonometry with central corneal thickness) was still found to have high in nine patients.
- All the nine patients had suspicious optic disc on fundus examination.
- All the nine patients showed depression in the fields by Octopus Automated perimetry.
- All the nine patients showed open angle on Goldmann 's single mirror gonioscopy.
- All the nine patients belong to Type II DM .
- All the nine patients were above the age of 40 years.
- All the nine patients had DM of > 5 years of duration.

- Among the total of 200 patients in our study , Nine patients (4.5 %) were diagnosed to have glaucoma of open angle primary type . This correlates well with the previous studies supporting the role of diabetes in glaucoma of open angle primary type. Paul Mitschel *et al*.[95] in his study done on Australian population found out that the prevalence of glaucoma of open angle primary type was 5.5 % and in non – diabetics it was 2.8 % .

CONCLUSION

- Diabetic retinopathy is one of the important cause for Defective vision in diabetic patients .
- Glaucoma of primary open angle type is another asymptomatic condition affecting eye leading to irreversible visual loss .
- Both are common after 40 years of age .
- Both have polygenic inheritance .
- By the time glaucoma of primary open angle type is diagnosed, there is already some percentage loss of nerve fibres which is Irreversible .
- Screening should be done for all the diabetic patients for co-existing glaucomatous changes even if the patient is asymptomatic.
- This is very important in diabetic patients , as they have already compromised optic nerve head blood flow due to defects in auto-regulation mechanism .

- Hence it is mandatory to rule out early glaucomatous changes by measuring intra – ocular pressure , central corneal thickness , fundus examination for optic disc changes and automated perimetry for early field changes in all diabetic patients to prevent a potentially sight threatening condition .

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PROFORMA

NAME OF THE PATIENT :

AGE :

SEX :

OP No. :

Diabetes History :

1. Age of onset -
2. Duration(years) -

Past History :

1. H/O glaucoma -
2. Systemic Hypertension -
3. Thyroid problem -
4. Ocular trauma -
5. H/O treatment for ocular complaints -

Family History -

Family history of glaucoma -

Personal History

1. H/O smoking -
2. H/O alcohol consumption -

OCULAR EXAMINATION

			RE	LE
1.	Uncorrected Visual Acuity (UCVA)	:		
2.	Refraction	SPH :		
		CYL :		
		AXIS :		
3.	Best Corrected Visual Acuity (BCVA)	:		
4.	Non Contact Tonometry (mmHg)	:		
5.	Torch light Examination			
	EYE LIDS	:		
	CONJUNCTIVA	:		
	CORNEA	:		
	ANTERIOR CHAMBER	:		
	IRIS	:		
	PUPIL	:		
	LENS	:		

The above findings are confirmed with slit lamp examination .

6. Fundus examination done after pupillary dilatation (0.8 % tropicamide and 5 % phenylephrine)

Direct ophthalmoscopy :

The findings are confirmed with + 90 D and indirect ophthalmoscopy

In patients suspected to have signs of development of glaucoma , the following investigations are done :

RE LE

1. Applanation tonometry

2. Corrected IOP by Pachymetry

3. Angle study by Gonioscopy

4. Fundus Photo for documentation

5. Fields by Automated perimetry.

CONSENT FORM

I am Dr. K. NITHYA, carrying out a study on the topic **"SCREENING OF DIABETIC PATIENTS ATTENDING DIABETOLOGY OUT PATIENT DEPARTMENT FOR PRIMARY OPEN ANGLE GLAUCOMA".**

My research project is being carried out under the Department of Obstetrics and Gynaecology, Coimbatore Medical College and Hospital.

RESEARCH BEING DONE:

"**SCREENING OF DIABETIC PATIENTS ATTENDING DIABETOLOGY OUT PATIENT DEPARTMENT FOR PRIMARY OPEN ANGLE GLAUCOMA**".

SAMPLE SIZE:

200 patients.

STUDY PARTICIPANTS:

Diabetics patients attending Diabetology Out Patient Department, Coimbatore Medical College and Hospital, Coimbatore.

LOCATION:

CMCH, Coimbatore.

You, Shri./ Smt./ Kum. _____, aged ____ years, S/o / D/o / W/o _____, residing at _____ _____ are requested to be a participant in the research study **titled "SCREENING OF DIABETIC PATIENTS ATTENDING DIABETOLOGY OUT PATIENT DEPARTMENT FOR PRIMARY OPEN ANGLE GLAUCOMA"** in Government Medical College Hospital, Coimbatore. You satisfy eligibility criteria as per the inclusion criteria. You can ask any question or seek any clarifications on the study that you may have before agreeing to participate.

DECLINE FROM PARTICIPATION

You are hereby made aware that participation in this study is purely voluntary and honorary and that you have the option and the right to decline from participation in the study.

PRIVACY AND CONFIDENTIALITY

You are hereby assured about your privacy. Privacy of subject will be respected and any information about you or provided by you during the study will be kept strictly confidential.

AUTHORIZATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy be breached.

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by Dr. K. Nithya. I have read and understood the consent form / or it has been read and explained to me in my own language. The study has been fully explained to me, and I may ask questions at any time.

Signature / Left Thumb Impression of the Volunteer Date:

Place:

Signature and Name of witness Date:

Place:

Signature of the investigator:

Name of the investigator:

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் கண் மருத்துவ துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவி **திருமதி. க. நித்யா** அவர்கள் மேற்கொள்ளும் **நீரிழிவு புறநோயாளிகள் பிரிவில் சிகிச்சைக்கு வரும் நீரிழிவு நோயாளிகளுக்கு கண் நீர் அழுத்த நோயின் ஆரம்ப நிலை அறிகுறிகளை கண்டறிதல்** பற்றிய ஆய்வில் செய்முறை மற்றும் அனைத்து விளக்கங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெரிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னைப் பற்றிய அனைத்து விபரங்கள் பாதுகாக்கப் படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபணை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம்

தேதி

கையொப்பம் , ரேகை

KEY TO MASTER CHART

Sl No	-	Serial Number
OP No	-	Out Patient Number
UCVA	-	Uncorrected Visual Acuity
BCVA	-	Best Corrected Visual Acuity
NCT	-	Non – Contact Tonometry
AT	-	Applanation Tonometry
CCT	-	Central Corneal Thickness
IOP	-	Intra Ocular pressure
DM	-	Diabetes Mellitus
I	-	Type I Diabetes Mellitus
II	-	Type II Diabetes Mellitus
Open	-	Open angles on Gonioscopy
N	-	Normal optic disc
S	-	Suspicious optic disc

MASTER CHART

Sl. No.	OP No.	Name	Age	Sex	UCVA		BCVA		NCT		AT		CCT		Corrected IOP		Gonioscopy		Automated perimetry		Fundus		Type Of	Duration
					RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	DM	Of DM
1	1234	Sudhakar	49	M	6/9	6/9	6/6	6/6	16	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
2	1260	murugesh	47	M	6/6	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	6
3	1349	Arun	43	M	6/6	6/6	6/6	6/6	16	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
4	1397	Sivakami	48	F	6/6	6/6	6/6	6/6	14	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
5	1416	Ammasai	54	M	6/6	6/6	6/6	6/6	16	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
6	1438	Marappan	56	M	6/9	6/9	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
7	1454	subbayee	54	F	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
8	1471	Palanisamy	55	M	6/9	6/9	6/6	6/6	18	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
9	1532	Kuppan	55	M	6/6	6/9	6/6	6/6	15	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4
10	1556	Thulsiyamma	54	F	6/9	6/6	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
11	1558	sarojini	50	F	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
12	1570	Arusamy	55	M	6/6	6/6	6/6	6/6	24	25	24	24	552	556	24	23	open	open	Depression	Depression	S	S	2	8
13	1572	Mohammad	55	M	6/9	6/6	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
14	1575	Arumugam	50	M	6/6	6/9	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
15	1580	Saraswathy	55	F	6/6	6/6	6/6	6/6	16	13	-	-	-	-	-	-	-	-	-	-	N	N	2	5
16	1590	Jayanthi	47	F	6/9	6/6	6/6	6/6	15	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
17	1595	Nehru	54	M	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
18	1650	Sundarum	55	M	6/6	6/6	6/6	6/6	13	15	-	-	-	-	-	-	-	-	-	-	N	N	2	6
19	1770	Mallikai	54	F	6/9	6/6	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4
20	1775	Parvathy	49	F	6/6	6/6	6/6	6/6	19	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
21	1780	Velan	47	M	6/6	6/6	6/6	6/6	11	11	-	-	-	-	-	-	-	-	-	-	N	N	2	6
22	1786	Thulasi	51	F	6/6	6/6	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
23	1799	Lingan	47	M	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
24	1810	Thenmozhi	50	F	6/6	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
25	1850	Sree	50	F	6/6	6/6	6/6	6/6	25	25	24	24	554	552	24	24	open	open	Depression	Depression	S	S	2	6
26	1880	Fathima	47	F	6/6	6/6	6/6	6/6	18	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
27	1900	Tamilarasu	47	M	6/6	6/9	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	7
28	1950	Murugesan	46	M	6/6	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	4
29	1990	Murugan	49	M	6/6	6/9	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
30	1999	Chitra	51	F	6/6	6/6	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
31	2009	Dhana	45	F	6/6	6/6	6/6	6/6	16	14	-	-	-	-	-	-	-	-	-	-	N	N	2	4
32	2050	Molli	48	M	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
33	2090	Rajangam	52	M	6/6	6/9	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
34	2100	Nagaraj	44	M	6/9	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	6
35	2150	Selvan	49	M	6/6	6/6	6/6	6/6	15	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4
36	2199	Ushan	45	M	6/6	6/6	6/6	6/6	11	11	-	-	-	-	-	-	-	-	-	-	N	N	2	5
37	2255	Vijaya	46	F	6/6	6/6	6/6	6/6	14	14	-	-	-	-	-	-	-	-	-	-	N	N	2	6
38	2270	Chandran	47	M	6/6	6/6	6/6	6/6	16	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4

39	2290	Mariya	49	F	6/6	6/9	6/6	6/6	16	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
40	2356	Thangaraj	46	M	6/9	6/6	6/6	6/6	16	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
41	2558	Jamela	49	F	6/6	6/6	6/6	6/6	16	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
42	2667	Rajagopal	45	M	6/6	6/6	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
43	3000	Gracemary	44	F	6/6	6/6	6/6	6/6	12	12	-	-	-	-	-	-	-	-	-	-	N	N	2	5
44	3111	Nagan	46	M	6/6	6/9	6/6	6/6	14	11	-	-	-	-	-	-	-	-	-	-	N	N	2	6
45	3223	Alamelu	45	F	6/6	6/6	6/6	6/6	13	13	-			-	-	-	-	-	-	-	N	N	2	4
46	3456	Devaki	47	F	6/9	6/6	6/6	6/6	17	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
47	3458	Sundar	44	M	6/6	6/6	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
48	3459	Sharvesh	47	M	6/6	6/9	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	6
49	3460	Aiamu	50	F	6/9	6/6	6/6	6/6	30	29	29	28	562	565	29	26	open	open	Depression	Depression	S	S	2	7
50	3462	Ragila banu	49	F	6/6	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
51	3463	Chinnasamy	46	M	6/6	6/9	6/6	6/6	15	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
52	3465	Krishnasamy	49	M	6/9	6/6	6/6	6/6	16	15	-	-	-	-	-	-	-	-	-	-	N	N	2	6
53	3467	Raju	47	M	6/6	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
54	3469	Krishnan	50	M	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
55	3471	Santhakumar	45	M	6/6	6/9	6/6	6/6	16	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
56	3474	Ferrous	49	F	6/9	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
57	3477	Manimegalai	47	F	6/6	6/9	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
58	3479	Sahadevan	49	M	6/6	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
59	3485	Uma	45	F	6/9	6/6	6/6	6/6	15	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
60	3489	Sudhakaran	44	M	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
61	3490	Guna	45	M	6/6	6/6	6/6	6/6	14	13	-	-	-	-	-	-	-	-	-	-	N	N	2	6
62	3492	Murugan	45	M	6/6	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	4
63	3497	Jaffer	45	M	6/6	6/6	6/6	6/6	13	13	-	-	-	-	-	-	-	-	-	-	N	N	2	5
64	3498	Devi	44	F	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	1	6
65	3499	Pooranum	50	F	6/6	6/6	6/6	6/6	16	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4
66	3500	Chellammal	45	F	6/9	6/6	6/6	6/6	15	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
67	3506	Amruthan	49	M	6/6	6/6	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
68	3509	Sankar	45	M	6/6	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
69	3510	Rajaram	44	M	6/6	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
70	3511	Vasanthan	47	M	6/6	6/9	6/6	6/6	16	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
71	3521	Rajamma	47	F	6/6	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
72	3541	Ravi	48	M	6/6	6/6	6/6	6/6	13	12	-	-	-	-	-	-	-	-	-	-	N	N	2	5
73	3546	Palani	51	M	6/6	6/9	6/6	6/6	16	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
74	3556	Rajammal	47	F	6/9	6/6	6/6	6/6	22	22	22	21	575	572	19	19	Open	open	absent	absent	N	N	2	6
75	3567	Yasothammal	50	F	6/6	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
76	3578	Raja	45	M	6/6	6/6	6/6	6/6	13	12	-	-	-	-	-	-	-	-	-	-	N	N	2	6
77	3600	Valliyamma	46	F	6/6	6/6	6/6	6/6	15	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4
78	3605	Palani	47	M	6/6	6/6	6/6	6/6	16	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
79	3612	Nagalakshmi	47	F	6/6	6/6	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	6
80	3623	Abdul	46	M	6/6	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
81	3634	Gunasekar	45	M	6/6	6/9	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5

82	3645	Gopal	47	M	6/9	6/6	6/6	6/6	16	14	-	-	-	-	-	-	-	-	-	-	N	N	2	4
83	3665	Chandramohan	50	M	6/6	6/6	6/6	6/6	16	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
84	3670	Manoharan	44	M	6/6	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	4
85	3675	Kandhaiya	47	M	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
86	3680	Ganesan	55	M	6/9	6/6	6/6	6/6	25	24	24	23	550	545	24	23	open	open	present	present	S	S	2	8
87	3689	VeeraRagavan	55	M	6/6	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
88	3701	Selvaraj	55	M	6/6	6/6	6/6	6/6	16	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
89	3708	Palanisamy	53	M	6/6	6/6	6/6	6/6	15	14	-	-	-	-	-	-	-	-	-	-	N	N	2	4
90	3712	Chinnakannu	60	F	6/6	6/6	6/6	6/6	16	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
91	3723	Rajathi	46	F	6/6	6/9	6/6	6/6	18	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
92	3734	Lakshman	48	M	6/9	6/6	6/6	6/6	16	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
93	3745	Rangasamy	52	M	6/6	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4
94	3756	Selvi	50	F	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4
95	3765	Devaraj	45	M	6/6	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
96	3778	Selvan	50	M	6/9	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
97	3809	Soundamma	52	F	6/6	6/6	6/6	6/6	18	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
98	3812	Velusamy	52	M	6/6	6/6	6/6	6/6	25	25	24	25	552	560	24	24	open	open	Depression	Depression	S	S	2	7
99	3834	Mariya	47	F	6/6	6/6	6/6	6/6	17	17	-	-	-	-	-	-	-	-	-	-	N	N	1	5
100	3867	Palaniyamma	50	F	6/6	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
101	3875	Subramani	49	M	6/6	6/9	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	4
102	3879	Alamu	45	F	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	5
103	3880	Kala	47	F	6/9	6/6	6/6	6/6	14	15	-	-	-	-	-	-	-	-	-	-	N	N	2	6
104	3990	Thangamuthu	45	M	6/6	6/6	6/6	6/6	17	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
105	3996	Indira	48	F	6/6	6/9	6/6	6/6	18	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4
106	3997	Selvam	46	M	6/6	6/6	6/6	6/6	14	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
107	4000	Tamilselvi	52	F	6/9	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
108	4112	Aadhi	44	M	6/6	6/6	6/6	6/6	14	13	-	-	-	-	-	-	-	-	-	-	N	N	2	4
109	4213	Philomina	50	F	6/6	6/6	6/6	6/6	15	13	-	-	-	-	-	-	-	-	-	-	N	N	2	5
110	4321	Nanjan	48	M	6/6	6/6	6/6	6/6	18	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
111	4567	Mary	51	F	6/6	6/6	6/6	6/6	25	26	25	25	560	556	24	24	open	open	Depression	Depression	S	S	2	8
112	5111	Maran	60	M	6/9	6/6	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
113	5123	Sulochana	58	F	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
114	5345	Devaki	57	F	6/6	6/9	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	4
115	6011	Rajeswari	50	F	6/6	6/6	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
116	6099	Thulasiram	44	M	6/6	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	6
117	6111	Sreedharan	55	M	6/6	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
118	6210	Kaveri	52	F	6/6	6/9	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
119	6321	Mariyappan	54	M	6/6	6/6	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
120	6432	Dhanabakyam	56	F	6/9	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	4
121	6543	Chitra	44	F	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
122	7112	Manikam	53	M	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
123	7219	Devi	55	F	6/6	6/9	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
124	7321	Malar	49	F	6/6	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4

125	7407	Thangam	47	F	6/9	6/6	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
126	7412	Mannar	57	M	6/9	6/9	6/6	6/6	26	24	25	25	550	552	25	25	open	open	Depression	Depression	S	S	2	12
127	7432	Babu	47	M	6/6	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4
128	7543	Bama	48	F	6/6	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
129	7654	Neela	47	F	6/6	6/6	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	6
130	7756	Balaji	50	M	6/6	6/9	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
131	7856	Muni	52	M	6/6	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	4
132	7965	Meenakshi	49	F	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
133	8000	Flora	52	F	6/6	6/9	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4
134	8122	Durai	51	M	6/6	6/6	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
135	8233	Mani	49	M	6/9	6/6	6/6	6/6	19	19	-	-	-	-	-	-	-	-	-	-	N	N	2	6
136	8344	Kala	57	F	6/6	6/6	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
137	8400	Nambi	52	M	6/6	6/9	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4
138	8599	Kaali	49	M	6/6	6/6	6/6	6/6	22	22	21	21	566	565	19	19	open	open	Normal	Normal	N	N	2	8
139	8612	Kannan	50	M	6/6	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
140	8622	Lalitha	52	F	6/6	6/6	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
141	8633	Kavitha	49	F	6/9	6/6	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
142	8655	Raju	47	M	6/6	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	6
143	8677	Panner	49	M	6/6	6/9	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
144	8709	Janaki	53	F	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	4
145	8712	Rathinum	47	M	6/6	6/6	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
146	8723	Balan	50	M	6/6	6/9	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	6
147	8734	Muruga	49	M	6/9	6/6	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	1	5
148	8745	Pangajam	52	F	6/6	6/6	6/6	6/6	22	21	21	21	572	575	18	18	open	open	Normal	Normal	N	N	2	6
149	8812	Parimala	49	F	6/6	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
150	8823	Anghamuthu	47	M	6/6	6/9	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	6
151	8845	Naren	45	M	6/6	6/6	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
152	8867	Inban	49	M	6/6	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	1	5
153	8912	Kamalam	52	F	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
154	8934	Vasantha	50	F	6/9	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
155	8999	Selvan	52	M	6/6	6/6	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	6
156	9012	Jamuna	47	F	6/6	6/9	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
157	9032	Manoj	49	M	6/9	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	6
158	9111	Suresh	51	M	6/6	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
159	9221	Pappu	47	F	6/6	6/6	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	6
160	9321	Dhanraj	51	M	6/6	6/6	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
161	9423	Banu	56	F	6/6	6/9	6/6	6/6	25	26	25	25	556	560	24	24	open	open	Depression	Depression	S	S	2	11
162	9455	Stephen	49	M	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
163	9500	Moorthy	53	M	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
164	9645	Antony	51	M	6/9	6/6	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
165	9700	Sarala	55	F	6/6	6/6	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
166	9709	Velu	49	M	6/6	6/6	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
167	9712	Balraj	50	M	6/6	6/9	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4

168	9723	Kalyani	47	F	6/6	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
169	9734	Devainai	46	F	6/9	6/6	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	6
170	9745	Eswaran	52	M	6/6	6/6	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
171	9756	Grish	49	M	6/6	6/9	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	6
172	9812	Arun	52	M	6/6	6/6	6/6	6/6	22	23	21	22	572	575	19	19	open	open	Normal	Normal	N	N	2	7
173	9834	Bakyam	49	F	6/6	6/6	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	4
174	9856	Jyothi	48	F	6/9	6/6	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
175	9867	Ram	51	M	6/6	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	6
176	9904	Maniyan	45	M	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	5
177	9912	Saravanan	50	M	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
178	9921	Padmanaban	52	M	6/6	6/6	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
179	9923	Karuppan	49	M	6/9	6/6	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
180	9934	Govindhan	47	M	6/6	6/6	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
181	9945	Valli	48	F	6/6	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	5
182	9967	Vargeez	49	M	6/6	6/9	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	6
183	10001	Aarathan	52	M	6/6	6/6	6/6	6/6	25	24	24	24	550	545	24	24	open	open	Depression	Depression	S	S	2	8
184	10014	Helarani	47	F	6/6	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4
185	10021	Manjula	53	F	6/9	6/6	6/6	6/6	19	18	-	-	-	-	-	-	-	-	-	-	N	N	2	5
186	10034	Veeran	55	M	6/6	6/6	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	6
187	10056	Ruckmani	47	F	6/6	6/6	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
188	10078	Krishana	53	M	6/6	6/9	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	6
189	10104	Sabeer	48	M	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	4
190	10114	Arjunan	49	M	6/6	6/9	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5
191	10213	Gomathi	50	F	6/6	6/6	6/6	6/6	18	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
192	10223	Muthu	47	M	6/6	6/6	6/6	6/6	12	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
193	10245	Hari	45	M	6/6	6/9	6/6	6/6	15	16	-	-	-	-	-	-	-	-	-	-	N	N	2	6
194	10277	Abith	47	M	6/9	6/6	6/6	6/6	18	15	-	-	-	-	-	-	-	-	-	-	N	N	2	4
195	10309	Kokila	52	F	6/6	6/6	6/6	6/6	22	22	21	21	565	570	19	19	open	open	Normal	Normal	N	N	2	8
196	10312	samy	48	M	6/6	6/9	6/6	6/6	17	19	-	-	-	-	-	-	-	-	-	-	N	N	2	6
197	10322	Munusamy	47	M	6/6	6/6	6/6	6/6	18	14	-	-	-	-	-	-	-	-	-	-	N	N	2	5
198	10344	Balan	50	M	6/6	6/6	6/6	6/6	17	18	-	-	-	-	-	-	-	-	-	-	N	N	2	6
199	10453	Karuppan	52	M	6/6	6/6	6/6	6/6	19	17	-	-	-	-	-	-	-	-	-	-	N	N	2	6
200	10555	Mariyamma	49	F	6/6	6/6	6/6	6/6	17	16	-	-	-	-	-	-	-	-	-	-	N	N	2	5